BIOCHEMICAL ABNORMALITIES IN NEONATAL SEIZURES: A CASE-CONTROL STUDY FROM A TERTIARY CARE CENTRE

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ABSTRACT
Background: Neonatal seizures represent one of the most common neurological emergencies in newborns, with a frequency of 0.95–3.5 per 1000 live births. Biochemical disturbances including hypoglycaemia, hypocalcaemia, hyponatraemia and hyperbilirubinaemia are important and treatable causes. Early recognition is critical to reduce neurodevelopmental morbidity.
Objective: To study and correlate biochemical abnormalities in neonatal seizures in a tertiary care centre using a case-control design.
Methods: A prospective case-control study was conducted over 24 months (September 2019 – September 2021) in the NICU of MGM Medical College, Kishanganj. Fifty neonates with seizures (cases) and 50 healthy neonates without seizures (controls) were enrolled. Blood glucose, serum calcium, serum sodium and total serum bilirubin were measured and compared between groups using SPSS v21.0.
Results: Male predominance was observed in the case group (58% vs 48%). Mean blood glucose (58.30 ± 25.03 mg/dL vs 72.00 ± 4.14 mg/dL), serum calcium (6.94 ± 0.82 mg/dL vs 8.61 ± 0.42 mg/dL) and serum sodium (124.38 ± 11.83 mEq/L vs 135.40 ± 3.09 mEq/L) were significantly lower in cases, while serum bilirubin (8.48 ± 6.22 mg/dL vs 2.74 ± 0.69 mg/dL) was significantly higher. All differences were statistically significant ($p < 0.0001$).
Conclusion: Biochemical abnormalities—particularly hypocalcaemia, hypoglycaemia, hyponatraemia and hyperbilirubinaemia—are important contributors to neonatal seizures. Prompt biochemical workup of all neonates presenting with seizures is essential for targeted management and improved neurological outcome.

Keywords: Neonatal Seizures, Hypoglycaemia, Hypocalcaemia, Hyponatraemia, Bilirubin Encephalopathy, Nicu.

INTRODUCTION
Neonatal seizures are defined as paroxysmal, involuntary disturbances of brain function occurring within the first four weeks of life in full-term infants, and up to 44 weeks corrected gestational age in premature infants.[1,2] They represent the most frequent neurological emergency in the neonatal period, with an overall incidence of 0.95–3.5 per 1000 live births.[3,4] Incidence rises dramatically in very low birth weight infants (birth weight <1500 g), reaching up to 57.5 per 1000.[5,6,7]
Seizures in neonates are almost invariably symptomatic of an underlying cerebral or metabolic disorder.

www.ajmrhs.com
eISSN: 2583-7761

Date Received: 22-05-2026
Date Accepted: 30-05-2026
Date of Publication: 29-06-2026

Hypoxic-ischaemic encephalopathy (HIE) is traditionally considered the most frequent cause; however, biochemical abnormalities—including hypoglycaemia, hypocalcaemia, hyponatraemia and hyperbilirubinaemia—are prevalent and potentially reversible aetiologies that may coexist with or occur independently of HIE.[3,8]
Biochemical disturbances may be primary or may arise as secondary complications of birth asphyxia, septicaemia, intracranial haemorrhage or meningitis. As neonatal sepsis and meningitis are well-recognized precipitants of metabolic derangements leading to seizures, microbiological evaluation forms an integral part of the diagnostic work-up in suspected neonatal seizures. Appropriate microbiological investigations help differentiate primary metabolic seizures from infection-associated biochemical abnormalities and guide timely antimicrobial therapy.[8,9] Hyponatraemia, for example, frequently accompanies brain damage from asphyxia or meningitis due to inappropriate secretion of antidiuretic hormone (SIADH) or

iatrogenic fluid overload.[10] Hypoglycaemia frequently complicates sepsis and asphyxia owing to increased glucose utilisation and impaired hepatic glucose mobilisation.[11] Early- and late-onset hypocalcaemia are recognised sequelae of perinatal hypoxia, prematurity, and high-phosphate formula feeding.[12,13]

Given that these biochemical disturbances are treatable and their early correction substantially alters prognosis, biochemical screening should be performed in every neonate presenting with seizures, irrespective of an apparent alternative cause.[3,8] The present study was undertaken to characterise the biochemical profile of neonatal seizures in a tertiary care centre and to compare key biochemical parameters between affected neonates and healthy controls.

Aims and Objectives

Aim: To study biochemical abnormalities in neonatal seizures in a tertiary care centre (case-control study).

Objectives:

1. To measure biochemical abnormalities (blood glucose, serum calcium, serum sodium, serum bilirubin) in neonates with seizures.
2. To correlate the biochemical aetiology with clinical features of neonatal seizures.

MATERIALS AND METHODS

Study Design and Setting

A prospective case-control study was conducted in the Neonatal Intensive Care Unit (NICU) and Paediatric Department, Mata Gujri Memorial Medical College and L.S.K. Hospital, Kishanganj, Bihar—a tertiary care centre—from September 2019 to September 2021 (24 months).

Study Participants

Cases: Fifty neonates admitted to the NICU with clinical features of seizures (paroxysmal involuntary motor activity, tonic posturing, clonic movements, subtle automatisms or apnoeic episodes with autonomic features) were enrolled. Diagnosis was confirmed by clinical examination, biochemical profiling and neuroimaging where indicated.

Controls: Fifty apparently healthy neonates admitted for reasons other than neurological illness were enrolled. Neonates with hepatic, muscular or cardiac disorders were excluded from the control group.

Cases were further classified as: (i) seizures due to hypoglycaemia, (ii) seizures due to hypocalcaemia, (iii) seizures due to hyponatraemia, (iv) bilirubin encephalopathy (kernicterus), and (v) seizures due to cerebral abnormality (HIE, intracranial haemorrhage). Cases of meningitis, intraventricular haemorrhage and subarachnoid haemorrhage confirmed on CSF analysis and neuroimaging were excluded to sharpen the biochemical conclusions.

Diagnostic Criteria

Hypoglycaemia: Blood glucose <25 mg/dL in the first 4 hours; <35 mg/dL between 4 and 24 hours of life, irrespective of gestational age or birth weight.[14]

Hypocalcaemia: Serum calcium <7.0 mg/dL (ionised calcium <4 mg/dL) during the first four weeks of life.[12,13]

Hyponatraemia: Serum sodium <120 mEq/L, in association with depleted, normal or expanded extracellular fluid volume.[10]

Hyperbilirubinaemia/Bilirubin encephalopathy: Serum bilirubin above age-specific thresholds; clinical features of kernicterus (lethargy, high-pitched cry, retrocollis, opisthotonus, setting sun sign, seizures).[15,16]

Microbiological Evaluation

All neonates presenting with seizures underwent clinical assessment for evidence of neonatal sepsis according to institutional NICU protocols. When clinically indicated, blood culture, cerebrospinal fluid (CSF) analysis including Gram stain and culture, and sepsis screening (complete blood count, C-reactive protein, and other relevant biomarkers) were performed before initiation of antimicrobial therapy wherever feasible. Neonates with microbiologically confirmed meningitis or culture-confirmed sepsis associated with central nervous system infection were excluded from the final biochemical analysis to minimise confounding from infection-related metabolic disturbances.

Laboratory Methods

Blood glucose: Enzymatic glucose oxidase–peroxidase (GOD-POD) colorimetric method; absorbance measured at 505 nm. Blood collected in fluoride oxalate vials.[17]

Serum calcium: Photometric Arsenazo III method; magnesium interference eliminated by 8-hydroxyquinoline-5-sulphonic acid; wavelength 650 nm.[18]

Serum sodium: Direct ion-selective electrode (ISE) method using a Na+/K+ analyser; detection range 80–200 mmol/L.[19]

Serum bilirubin: Van den Bergh diazo reaction (Jendrassik–Grof modification); total and direct fractions determined at 540 nm; indirect bilirubin calculated by difference.[20]

Blood cultures were processed using standard microbiological techniques following institutional laboratory protocols. CSF specimens, when indicated, were subjected to routine cytological, biochemical, Gram stain and culture examination according to standard microbiological procedures.

Statistical Analysis

All data were recorded on a pre-structured proforma. Continuous variables were expressed as mean ± standard deviation (SD). Group comparisons were performed using the independent samples t-test. A p-value <0.05 was considered statistically significant. Statistical analysis was conducted using SPSS version 21.0 (IBM Corp., Chicago, USA).

Ethical Approval

The study protocol was approved by the Institutional Ethics Committee of Mata Gujri Memorial Medical College. Written informed consent was obtained from parents or legal guardians of all enrolled neonates in their vernacular language prior to enrolment.

RESULTS

Demographic Profile

A total of 100 neonates were studied: 50 cases and 50 controls. Neonates with microbiologically confirmed meningitis or culture-confirmed central nervous system infection identified during the diagnostic work-up were excluded from the final biochemical analysis in accordance with the predefined exclusion criteria. In the case group, 29 (58%) were male and 21 (42%) were female (male:female ratio 1.38:1). In the control group, 24 (48%) were male and 26 (52%) were female (Table 1). The age of cases ranged from 0 to 27 days (mean 8.3 days). The majority of seizures (66%) occurred within the first 10 days of life.

Table 1. Age and sex distribution of cases and controls

Age (days)	Case Males	Case Females	Case Total	Control Males	Control Females
0–5	14 (28%)	05 (10%)	19 (38%)	10 (20%)	12 (24%)
6–10	05 (10%)	09 (18%)	14 (28%)	07 (14%)	09 (18%)
11–15	08 (16%)	06 (12%)	14 (28%)	05 (10%)	04 (8%)
16–21	01 (2%)	00 (0%)	01 (2%)	01 (2%)	01 (2%)
22–27	01 (2%)	01 (2%)	02 (4%)	01 (2%)	00 (0%)
Total	**29 (58%)**	**21 (42%)**	**50 (100%)**	**24 (48%)**	**26 (52%)**

Biochemical Findings

All four biochemical parameters showed statistically significant differences between cases and controls ($p < 0.0001$). The results are summarised in Table 2.

Table 2. Comparison of biochemical parameters between cases and controls

Parameter	Cases Mean ± SD	Controls Mean ± SD	Case Range	Control Range	p-value
Blood Glucose (mg/dL)	58.30 ± 25.03	72.00 ± 4.14	25–120	60–90	<0.0001
Serum Calcium (mg/dL)	6.94 ± 0.82	8.61 ± 0.42	5–8	8–10.5	<0.0001
Serum Sodium (mEq/L)	124.38 ± 11.83	135.40 ± 3.09	100–142	130–142	<0.0001
Serum Bilirubin (mg/dL)	8.48 ± 6.22	2.74 ± 0.69	2–28	1.2–6	<0.0001

Blood Glucose

The mean blood glucose level in cases was 58.30 ± 25.03 mg/dL compared with 72.00 ± 4.14 mg/dL in controls ($p < 0.0001$). Blood glucose was the most variable parameter in the case group, reflecting heterogeneity in the degree of hypoglycaemia. This finding is consistent with previously reported series.[21,22,23,24,25]

Serum Calcium

Mean serum calcium was significantly lower in cases (6.94 ± 0.82 mg/dL) than in controls (8.61 ± 0.42 mg/dL; $p < 0.0001$). A serum calcium level below 7.0 mg/dL during the first four weeks of life was designated as neonatal hypocalcaemia. Hypocalcaemia was both early-onset (primarily due to prematurity and perinatal hypoxia) and late-onset (associated with high phosphate-content formula feeds).[12,13,26,27]

Serum Sodium

The mean serum sodium level was 124.38 ± 11.83 mEq/L in cases versus 135.40 ± 3.09 mEq/L in controls ($p < 0.0001$). Hyponatraemia (serum sodium <120 mEq/L) was attributed to SIADH secondary to perinatal asphyxia, meningitis or intraventricular haemorrhage, and to sodium depletion from gastrointestinal losses or renal salt-wasting.[10,28,29,30]

Serum Bilirubin

Total serum bilirubin was markedly elevated in cases (8.48 ± 6.22 mg/dL; range 2–28 mg/dL) compared with controls (2.74 ± 0.69 mg/dL; $p < 0.0001$). Bilirubin encephalopathy (kernicterus) was the underlying mechanism in neonates with severe hyperbilirubinaemia presenting with seizures, consistent with published literature.[15,16,31,32]

DISCUSSION

This case-control study evaluated four key biochemical parameters in 50 neonates with seizures and 50 healthy neonatal controls admitted to a tertiary care NICU. All parameters showed statistically significant differences, confirming that metabolic derangements are important and measurable contributors to neonatal seizures.

The blood glucose level was significantly lower in cases than in controls ($p < 0.0001$). Neonatal hypoglycaemia is multifactorial: blood glucose in neonates is physiologically 60–70% of the maternal level at birth and falls further during the first 24 hours.[14] Predisposing conditions include intrauterine growth restriction, prematurity, perinatal asphyxia, hypothermia, septicaemia and infants of diabetic mothers.[21,22] Our results align with those of Cornblath et al.[22,23], Koh et al.[24], and Lucas & Morley[25], all of whom documented reduced blood glucose in neonates with hypoglycaemic seizures. Although neonates with microbiologically confirmed meningitis and central nervous system infections were excluded from the final analysis, neonatal sepsis remains an important differential diagnosis in infants presenting with seizures. Systemic infection can precipitate hypoglycaemia, electrolyte disturbances, and hyperbilirubinaemia through increased metabolic demand, impaired hepatic glucose homeostasis, inflammatory responses, and syndrome of inappropriate antidiuretic hormone secretion (SIADH). Therefore, microbiological evaluation, including blood cultures and cerebrospinal fluid analysis when clinically indicated, complements biochemical investigations by distinguishing primary metabolic seizures from infection-associated metabolic derangements and facilitating timely antimicrobial therapy.

The significantly lower serum calcium in cases ($p < 0.0001$) reflects the multifaceted pathophysiology of neonatal hypocalcaemia: deficient calcium stores in preterm infants (most placental transfer of calcium occurs in the third trimester), delayed parathyroid response, reduced glomerular filtration rate with phosphate retention, and perinatal stress causing catabolism and bicarbonate therapy.[12,13,26,27] These observations are congruent with findings reported by Mirzahi et al.[26], O'Brien & Goodman[27], and Koo & Tsang.[13]

Hyponatraemia ($p < 0.0001$) in neonates with seizures was consistent with results of Pasi Rachna et al.[33], who found hyponatraemia in 59.2% of neonates with seizures and associated it with significantly higher mortality. In our series, hyponatraemia was most plausibly secondary to SIADH triggered by asphyxia, meningitis or intraventricular haemorrhage.[10,28,29,30] Our results are also consistent with those of Finberg et al.[28] and Feldman et al.[10]

Elevated serum bilirubin in cases ($p < 0.0001$) is in keeping with the well-established relationship between unconjugated hyperbilirubinaemia and bilirubin encephalopathy. Predisposing factors include Rh incompatibility, ABO incompatibility, G6PD deficiency, polycythaemia, septicaemia and occult haemorrhage.[15,16,31,32] Our findings parallel those of Singhal et al.[31], Narang et al.[32], and Stevenson et al.[34]

Collectively, these findings echo those of Arunkumar et al.[35] (2017), who reported one or more biochemical abnormalities in 82% of neonates with seizures, and Kamatham Madhusudhan et al.[36] (2016), who found hypoglycaemia as the most common primary abnormality in preterm infants and hyponatraemia as the most frequent secondary abnormality in term babies. Manoj D et al.[37] (2018) similarly concluded that biochemical abnormalities are common and often unrecognised, underscoring the need for routine metabolic screening in all neonates with seizures.

No significant correlation was found between any individual biochemical parameter and the other three parameters, suggesting that each disturbance contributes independently to seizure generation and warrants separate evaluation and correction.

Limitations and Future Directions

This study has certain limitations. First, it was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalizability of the findings. Second, the biochemical evaluation was restricted to blood glucose, serum calcium, serum sodium, and serum bilirubin; other important metabolic parameters such as serum magnesium, phosphate, and lactate were not assessed. Third, although microbiological evaluation was performed in clinically suspected cases to exclude central nervous system infections, detailed microbiological findings were not analysed as part of the study objectives. Finally, the absence of long-term neurodevelopmental follow-up precluded assessment of the prognostic impact of the identified biochemical abnormalities.

Future multicentre studies with larger sample sizes should incorporate comprehensive metabolic and microbiological profiling along with neurophysiological and neuroimaging findings. Longitudinal follow-up of affected neonates is also warranted to evaluate the relationship between early biochemical abnormalities, infectious etiologies, treatment response, and long-term neurodevelopmental outcomes.

CONCLUSION

This case-control study demonstrated that neonates presenting with seizures had significantly lower blood glucose, serum calcium, and serum sodium levels, along with significantly higher serum bilirubin levels, compared with healthy controls. These findings reaffirm that hypoglycaemia, hypocalcaemia, hyponatraemia, and hyperbilirubinaemia are important and potentially reversible biochemical abnormalities associated with neonatal seizures.

Early identification and prompt correction of these metabolic disturbances are essential to improve clinical outcomes and reduce the risk of long-term neurological sequelae. In addition to routine biochemical evaluation, appropriate microbiological assessment in clinically suspected cases is valuable for distinguishing primary metabolic seizures from infection-associated metabolic derangements and guiding timely antimicrobial therapy. A multidisciplinary diagnostic approach integrating clinical, biochemical, and microbiological investigations can facilitate early diagnosis, targeted management, and improved neonatal outcomes.

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How to cite this article: Dr. Kumar Varuneshwar, Dr. Rahul Kumar, Dr. Sonam Sharma, BIOCHEMICAL ABNORMALITIES IN NEONATAL SEIZURES: A CASE-CONTROL STUDY FROM A TERTIARY CARE CENTRE, Asian J. Med. Res. Health Sci., 2026; 4 (2):- 1613-1618.
Source of Support: Nil, Conflicts of Interest: None declared.